AMENDMENTS TO THE CLAIMS

1. (Previously Presented) A method of diagnosing a cardiovascular condition characterized by increased expression of a Fit-1/ST2 nucleic acid molecule or an expression product thereof, said method comprising:

a) contacting a biological sample from a subject with an agent, wherein said agent specifically binds to said Fit-1/ST2 nucleic acid molecule, an expression product thereof, or a fragment of an expression product thereof; and

b) measuring the amount of bound agent and determining therefrom if the expression of said Fit-1/ST2 nucleic acid molecule or of an expression product thereof is increased relative to a predetermined value, wherein the expression increased relative to a predetermined value is diagnostic of the condition.

2.-5. (Canceled)

6. (Previously Presented) The method of claim 1, wherein the cardiovascular condition is selected from the group consisting of myocardial infarction, stroke, arteriosclerosis, and heart failure.

7. (Previously Presented) The method of claim 1, wherein the cardiovascular condition is cardiac hypertrophy.

8. (Currently amended) A method for monitoring a sample of a patient having or suspected of having cardiovascular condition ~~determining regression, progression or onset of a cardiovascular condition in a subject characterized by increased expression of a nucleic acid molecule or an expression product thereof~~, comprising:

assaying ~~monitoring~~ a sample from [[a]] the patient[[,]] for increased expression relative to a predetermined value of any of

(i) a Fit-1/ST2 nucleic acid molecule,

(ii) a polypeptide encoded by the nucleic acid of part (i), or

(iii) a peptide fragment of the polypeptide of part (ii).[[,]]

~~as a determination of the regression, progression or onset of said cardiovascular condition in the subject~~.

9. (Canceled)

10. (Previously Presented) The method of claim 8, wherein the step of monitoring comprises contacting the sample with a detectable agent selected from the group consisting of:

(a) an isolated nucleic acid molecule which hybridizes to the nucleic acid molecule of part (i), and

(b) an antibody or an antigen binding fragment thereof which binds the polypeptide of part (ii), or the peptide of part (iii),

11.-36. (Canceled)

37. (Previously Presented) The method of claim 1, wherein the sample is a biological fluid or a tissue.

38. (Previously Presented) The method of claim 1, wherein the biological fluid is blood or serum.

39. (Previously Presented) The method of claim 1, wherein the agent is (i) an isolated nucleic acid molecule that hybridizes to the Fit-1/ST2 nucleic acid molecule or (ii) an antibody that binds the polypeptide encoded by the Fit-1/ST2 nucleic acid molecule, or an antigen-binding fragment of the antibody.

40. (Previously Presented) The method of claim 39, wherein the nucleic acid or the antibody is labeled with a radioactive label or an enzyme.

41. (Previously Presented) The method of claim 1, wherein the cardiovascular condition is characterized by mechanical strain, mechanical overload or mechanically-induced deformation in cardiac cells or tissue.

42. (Previously Presented) The method of claim 8, wherein the sample is a biological fluid or a tissue.

43. (Previously Presented) The method of claim 8, wherein the biological fluid is blood or serum.

44. (Previously Presented) The method of claim 10, wherein the nucleic acid of part (a) or the antibody of part (b) is labeled with a radioactive label or an enzyme.

45. (Previously Presented) The method of claim 10, comprising assaying the sample for the peptide of part (iii).

46. (Previously Presented) The method of claim 8, wherein the cardiovascular condition is selected from the group consisting of myocardial infarction, stroke, arteriosclerosis, and heart failure.

47. (Previously Presented) The method of claim 8, wherein the cardiovascular condition is cardiac hypertrophy.

48. (Previously Presented) The method of claim 8, wherein the cardiovascular condition is characterized by mechanical strain, mechanical overload or mechanically-induced deformation in cardiac cells or tissue.